# Cognitive Control Deficits in Individuals with Differing Levels of Autistic Traits

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**Abstract**

Deficits in inhibitory control are common in Autism Spectrum Disorders (ASD) and are associated with higher levels of repetitive behaviours. Inhibitory deficits may present as an inability to stop a prepotent motor response (reactive inhibition), or as an inability to delay a response onset before it is performed (proactive inhibition). Previous studies have found conflicting results in reactive inhibition deficits in children with ASD indicating heterogeneity in stopping ability, while limited research into proactive inhibition has demonstrated more consistent deficits. This study aims to explore deficits in both types of inhibition in individuals from the general population with differing levels of autistic traits, by comparing two tasks measuring proactive and reactive inhibition. A Stop Signal Task (SST) and reinforcement learning task were administered to 152 participants (18-81 years). Level of autistic traits was measured using the AQ-28 scale. Stop Signal Reaction Time (SSRT) (an index of reactive inhibition) and post-error slowing (a measure of proactive inhibition) were examined in the SST, while another measure of proactive inhibition (reaction time between trials of high and low conflict) was obtained from the reinforcement learning task. Results indicated no significant deficits in both reactive and proactive inhibition regardless of self-reported autistic trait level. A modest interaction effect between age and SSRT predicted Routine subscale score on the AQ-28, suggesting that repetitive behaviour level can be altered by reactive inhibition ability changes across the lifespan. Cognitive control deficits in ASD therefore may be related to factors outside of response inhibition alone.

*Keywords:* autism, proactive, reactive, inhibition, cognitive control

## Declaration

This thesis contains no material which has been accepted for the award of any other degree or diploma in any University, and, to the best of my knowledge, this thesis contains no material previously published except where due reference is made. I give permission for the digital version of this thesis to be made available on the web, via the University of Adelaide's digital thesis repository, the Library Search and through web search engines, unless permission has been granted by the School to restrict access for a period of time.

Susan Giles

27th September 2021

## Contribution Statement

In writing this thesis, my supervisor and I collaborated to create the thesis aims and design of the methods of research. I was solely involved in conducting the literature search and selecting the proposed hypotheses of the study. I collaborated with other students in the Cognitive Neural Sciences Laboratory to collect all data used in this thesis, and was responsible for assisting with recruitment and participant testing. Ethics approval for this study was previously arranged by my supervisor prior to study commencement. My supervisor and I worked together when coding all analyses in R. I was solely involved in writing all aspects of this thesis, with the exception of the scripts that are supplied in the appendices.

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Secondly, I would like to thank the other students involved in the Cognitive Neural Sciences Laboratory, for their assistance in training me in data collection for this project. I would especially like to thank Brittany and Lauren for being so incredibly accommodating during this time, and for all of their hard work in designing and executing the lab protocols for this project. It was a wonderful experience working together and learning many useful research skills, and to have many memorable conversations and commiserations along the way.

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**Cognitive Control Deficits in Individuals with Differing Levels of Autistic Traits**

Autism Spectrum Disorder (ASD) is defined as 'a neurodevelopmental condition characterised by difficulties in social interaction and communication, as well as restricted or repetitive patterns of behaviour, interests or activities' (APA, 2013). Restricted and repetitive behaviours can be the most disabling features of autism (Bishop et al., 2007), and can cause stress on family members and carers. The executive function hypothesis of autism, an influential theory of the disorder, posits that deficits in cognitive control (flexibility, inhibitory control, attention shifting and working memory) are central to autistic symptom presentation (Bishop, 1993, Ozonoff, 1995, Pennington & Ozonoff, 1996, Russell, 1997, Hughes, 2001, Lopez et al., 2005), and may provide a potential explanation for the presence of repetitive behaviours. In particular, deficits in these executive functions may be driven by disruptions in frontostriatal circuitry, which can lead to the inability to suppress inappropriate actions (Christ et al., 2003), or cause repetition of over-learned behaviours (Solomon et al., 2008), contributing to the repetitive behaviours evident in individuals with ASD (Mosconi et al., 2009). This thesis will focus on two types of cognitive control, namely proactive and reactive control, utilising two tasks measuring response inhibition and reinforcement learning to estimate proactive and reactive control from behaviour. As proactive control is a type of cognitive control that has not yet been explored thoroughly in the context of ASD, this study therefore aims to address this gap by comparing autistic trait level and performance on proactive control measures. This would be potentially useful in understanding the underlying deficits in the function of frontostriatal circuitry in ASD, and may be useful in potentially identifying new targets for therapeutic intervention to lessen repetitive behaviours.

### 1.1 Cognitive Control

Cognitive control can be defined as the regulation of goal-directed, future-oriented and higher-order cognitive processes (Miller & Cohen, 2001). Cognitive control is thought to be a top-down process, meaning that behaviours performed by an individual need to be guided by internal motivations and goals, and is thought to be largely controlled by the prefrontal cortex (Miller & Cohen, 2001). Individuals can also perform behaviours that are reflexive or habitual in nature, and are referred to as automatic behaviours, or bottom-up processes (Miller & Cohen, 2001), and involve control via the basal ganglia (Solomon et al., 2011). Cognitive control is therefore often involved in overriding behaviours that have become reflexive or habitual if they are not appropriate in a given context. A key component of cognitive control involves proactive preparation for future events and focuses on goal-relevant information to bias attention, perception and action systems (Braver, 2012), while a second component reactively controls responses to incoming stimuli and addresses any conflicts that arise between systems to make necessary corrections (Braver, 2012). Proactive control is utilised in order to prepare to override future habitual automatic responses, whereas reactive control is involved in overriding an automatic response that is currently being performed. Proactive and reactive control can be measured in response inhibition and reinforcement learning tasks, which can be useful to assess to what extent these types of control are functioning effectively in different individuals.

### 1.2 Response Inhibition

Inhibition is the ability to cancel or suppress an action that is inappropriate or no longer contextually relevant. Inhibition has proactive and reactive components that involve overlapping frontostriatal pathways (Smittenaar et al., 2015). Reactive inhibition is the process of stopping a prepotent motor response when it is no longer appropriate (e.g.,

stopping suddenly at traffic lights when a car continues to come through an intersection), and is thought to be caused by the neural 'stop' signal reaching the thalamus before the motor command can be executed (Aron, 2011). Proactive inhibition is an adaptive cognitive control strategy that allows an individual to be prepared to stop a motor command in times of uncertainty (e.g., the ability to use contextual cues to prevent performing an inappropriate action), or after an error has been made previously (Aron, 2011). Reactive inhibition is thought to be more prominent in children, while proactive inhibition develops with maturity (Braver, 2012), typically around the age of 15 (Luna et al., 2007). Over the course of the lifespan, reactive inhibition declines with age, while proactive inhibition abilities remain constant once developed (Smittenaar et al., 2015). Response inhibition is measured using the Stop Signal Task (SST), as developed by Logan & Cowan (1984), and can measure both proactive and reactive inhibition. During the task, participants are required to respond to stimuli as fast as they can after presentation of a Go signal (e.g., pressing a corresponding button every time an arrow is shown), and cease the response when a Stop signal is shown in a minority of 'No-Go' trials. Reactive inhibition can be measured by calculating the Stop Signal Reaction Time (SSRT), which is the average reaction time on Go trials, minus the delay between the Go stimulus and the Stop stimulus that yields 50% of successful stops on No-Go trials (called the critical Stop Signal Delay). Proactive inhibition can be measured by post-error slowing that occurs when the participant makes an error on a No-Go trial, and subsequently slows down on the following Go trials, presumably out of caution to ensure better reactive inhibition on subsequent Stop trials. Both reactive and proactive inhibition will be assessed for this study, but proactive inhibition will be the key focus as it has not been explored thoroughly in the context of ASD.

### 1.3 Reinforcement Learning

Proactive control can also be assessed via reinforcement learning, which is described as the process by which an individual learns from probabilistic feedback on a trial-by-trial process (Sutton & Barto, 1998). Proactive control can be assessed by measuring proactive inhibition that occurs during the Probabilistic Selection Task, developed by Frank et al. (2004). During a training phase, participants are required to select between pairs of visual stimuli, and receive feedback on their choice as either being correct or incorrect in a probabilistic fashion. The participants' task therefore is to learn by trial-and-error which stimuli are more likely to receive positive feedback. In a subsequent testing phase, participants are then asked to select the most correct option without receiving feedback when pairs are mixed (i.e., when shown new stimulus combinations). Proactive inhibition occurs in the form of the participant slowing their reaction time for trials that have high conflict, meaning the two stimuli presented in the test trial had similar reinforcement probabilities (e.g., Stimulus A had an 80% chance of being correct during training, while Stimulus B had a 70% chance of being correct). Participants often have quicker reaction times for trials that have low conflict, meaning that the stimuli presented have significantly different and discernible reinforcement probabilities (e.g., Stimulus A had an 80% chance of being correct during training, while Stimulus B had a 20% chance of being correct). Reaction times between high and low conflict trials can be compared to assess levels of proactive inhibition. Participants also tend to slow their choices in order to increase their accuracy to avoid making mistakes or suboptimal choices (Cavanagh et al., 2014), which is why reaction time during high and low conflict trials can be used to assess proactive inhibition.

### 1.4 Summary of Measures

Both the reinforcement learning task and the SST engage habitual automatic responding by putting time pressure on participants to respond. Thus, high conflict trials on the reinforcement learning task and Stop trials in the SST engage the use of proactive control to ensure that the stimulus with greater probability of correct feedback is selected, or that errors are not made. The SST is also able to assess reactive inhibition via measuring the SSRT. The measures are summarised in Table 1.

**Table 1**

*Summary of Proactive and Reactive Inhibition Measures*

| | Proactive Inhibition | Reactive Inhibition |
|---|---|---|
| Stop Signal Task | **Post-Error Slowing:** The difference between Go trial reaction time after an error and Go trial reaction time before an error. | **Stop Signal Reaction Time:** The difference between Go trial reaction time and the critical Stop Signal Delay. |
| Reinforcement Learning Task | **High vs Low Conflict Responding:** The difference in reaction time between trials of high conflict (stimulus pairs with similar | **Not Measured.** |

reinforcement probabilities) and trials of low conflict (stimulus pairs with significantly different reinforcement probabilities).

### 1.5 Neurobiology of Cognitive Control

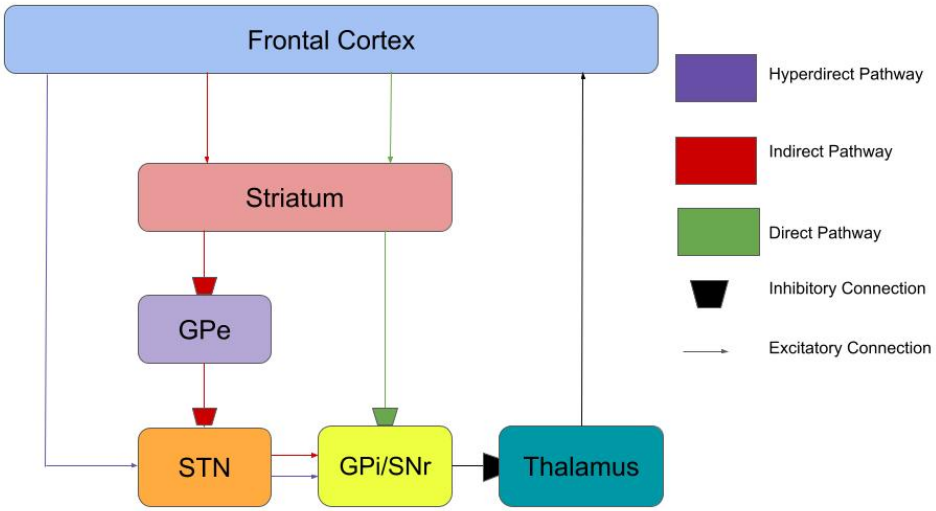
Neural circuits that are involved in cognitive control are affected in individuals with ASD. By understanding the basic neurobiology of these circuits involved in cognitive control, this knowledge can help us to make predictions about which type of cognitive control is likely to be compromised in the ASD population. The following section will briefly review these neural circuits that are known to be involved in cognitive control, and then the following section will subsequently review what we currently know about the functioning of these circuits in individuals with ASD.

Several components of the brain are involved in top-down cognitive control processes, including the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC) and the parietal cortex (Yarkoni et al., 2005). The DLPFC is involved in maintaining appropriate contexts for action (MacDonald et al., 2000). When a response conflict arises, the ACC acts as a detector, and signals the conflict to the DLPFC to allocate more control-related resources to figure out how to act (Egner & Hirsch, 2005). The parietal cortex is activated when it is necessary to switch attentional focus (Miller & Cohen, 2001). Proactive and reactive control signals travel from the frontal cortex to other brain areas, such as the basal ganglia, to

successfully change behaviours. Similar structures and pathways are implicated in both proactive and reactive control, in particular the subthalamic nucleus (STN), basal ganglia and the hyperdirect pathway, which functions as a pathway between the frontal cortex and the basal ganglia (see Figure 1). Basal ganglia functioning can be described through a 'centre-surround' model (Nambu et al., 2002). When a person is about to activate a voluntary movement, the hyperdirect pathway is activated and sends signals from the cortex to the STN, inhibiting large areas of the thalamus related to both the desired motor program and competing motor programs. Signals from the direct pathway then disinhibit areas of the thalamus that are related to the desired motor program only, while the indirect pathway then inhibits other targets extensively to ensure that the desired motor program ends when it should, and to ensure that no competing motor programs are activated (Nambu et al., 2002).

**Figure 1**

*Pathways Connecting the Frontal Lobe and Basal Ganglia*



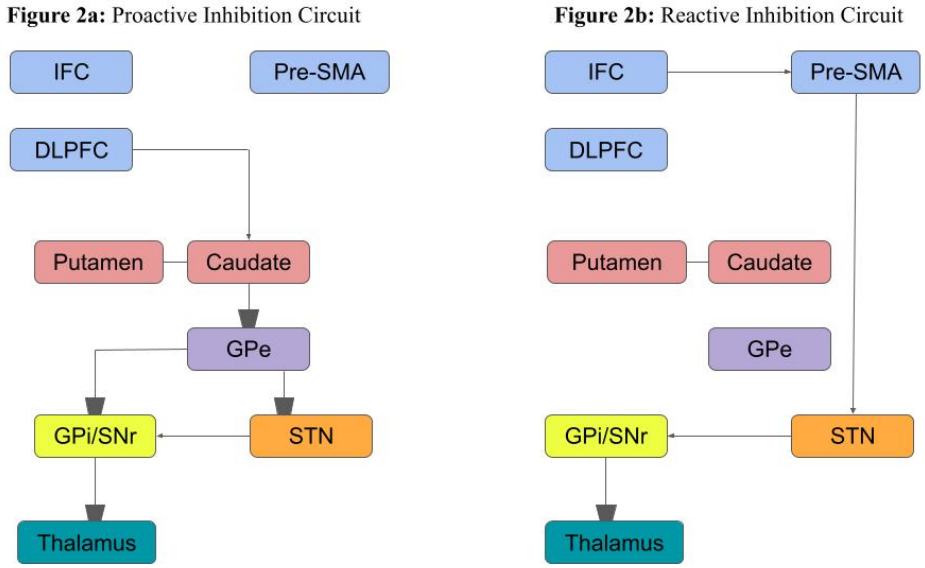

*Note:* The direct, indirect and hyperdirect cortico-basal ganglia-thalamo-cortical loops. The hyperdirect pathway involves excitatory signals sent from the frontal cortex via glutamatergic neurons to the subthalamic nucleus (STN). The STN then sends excitatory signals to the internal segment of the globus pallidus (GPi), which further inhibits the

thalamus, leading to weaker signals projecting back to the frontal cortex. The direct and indirect pathways both involve excitatory signals via glutamatergic neurons from the frontal cortex being projected to the striatum. The direct pathway sends inhibitory signals via GABAergic neurons to the GPi, which then disinhibits the thalamus, allowing excitatory signals to project back to the frontal cortex. This allows a planned motor command to be performed. The indirect pathway sends inhibitory signals via GABAergic neurons from the striatum to the external section of the globus pallidus (GPe), which inhibits the STN. Excitatory signals are then projected to the GPi which inhibits the thalamus further, preventing signals being projected back to the frontal cortex. When the hyperdirect and indirect pathways work together, only the selected motor program is performed and all competing programs are cancelled, while the direct pathway initiates the desired motor program (Nambu et al., 2002).

In the frontal cortex, the inferior frontal cortex (IFC) and pre-supplementary motor area (pre-SMA) are connected via neurons that make up the hyperdirect pathway (Aron & Poldrack, 2006). The hyperdirect pathway connects the IFC and the STN via a white matter tract (Aron et al., 2007), demonstrating the hyperdirect pathway functionally connects the frontal cortex and basal ganglia to execute and control voluntary movement. Similar neural pathways from the frontal cortex to the basal ganglia are activated for reactive and proactive inhibition (see Figure 2). Reactive inhibition involves the IFC, pre-SMA and the STN, which are components of the hyperdirect pathway (Aron, 2011, Jahanshahi et al., 2015). Proactive inhibition involves the DLPFC signaling the striatum, which signals to other components of the basal ganglia that belong to the indirect pathway (Aron, 2011, Jahanshahi et al., 2015).

**Figure 2**

*Proactive and Reactive Inhibition Circuits*




*Note:* Figure 2a illustrates the proactive inhibition pathway circuit. Signals from the dorsolateral prefrontal cortex (DLPFC) are sent through the indirect basal ganglia pathway, suggesting that the indirect pathway may mediate proactive inhibition (Jahanshahi et al., 2015). Figure 2b illustrates the reactive inhibition pathway. Signals from the inferior frontal cortex (IFC) and pre-supplementary motor area (pre-SMA) travel via the hyperdirect basal ganglia pathway.

In particular, the STN is involved in the switching between different types of inhibition (Aron & Poldrack, 2006, Ballanger et al., 2009, Benis et al., 2014). STN activity transiently increased for successful Stop trials in the SST in a healthy population, suggesting that hyperdirect pathway activation is involved in quickly stopping a motor response for a brief period of time, otherwise known as a reactive inhibition (Jahfari et al., 2019). A functional Magnetic Resonance Imaging (fMRI) study using a non-clinical population found that direct pathway activation occurs during Go trials during the SST, while IFC and STN activation occurred during Stop trials, indicating that STN activation blocked direct pathway signaling (Aron & Poldrack, 2006). Activation of the hyperdirect pathway was evident in

another fMRI study using the SST, with greater activation in the IFC, orbitofrontal cortex (OFC) and superior temporal gyrus in participants with ASD compared to controls (Chantiluke et al., 2015), showing higher levels of activation of the reactive inhibition circuit. To summarise, hyperdirect pathway activation and increased transient STN activity are involved in successfully stopping prepotent motor responses.

The STN is also involved in proactive inhibition, as it is involved in both the hyperdirect and indirect pathways. Previous research found that post-error slowing likely occurs via the same STN mechanism as outright response inhibition (Frank et al., 2006, Aron et al., 2007), via either the dynamic modulation of decision thresholds, or by an initial delay that precedes the decision-making process (Aron, 2011, Ratcliff & Frank, 2012). The STN is also the key basal ganglia component involved in the process of reducing premature responding, and therefore has a substantial effect on which stimulus is selected in a high conflict situation, especially when multiple motor programs are competing (Frank et al., 2006, Chikazoe et al., 2009, Benis et al., 2014). STN activity increases during decisions that have higher cognitive burden, suggesting there is a link between the STN and proactive control processes (Weingtraub & Zaghloul, 2013). STN activity between 2.5-5Hz has been found to contribute significantly to the length of post-error slowing, suggesting that the hyperdirect pathway is also involved in the mediation of proactive inhibition (Cavanagh et al., 2014), with differing levels of STN beta activity dissociating reactive and proactive inhibition (Benis et al., 2014). Slower inhibition times were associated with greater STN activity during high and low conflict trials in a reinforcement learning task in a non-clinical population (Jahfari et al., 2019). To summarise, the STN involves increased activity in both reactive and proactive inhibition scenarios, but this increase is transient for reactive inhibition and more prolonged for proactive inhibition events.

### 1.6 Neurobiological Changes in ASD

Structural and functional studies have found abnormalities in the frontal lobe in individuals with ASD, with the abnormalities correlating significantly with deficits in cognitive control and repetitive behaviour (Carper & Courchesne, 2000, 2005, Luna et al., 2002). In individuals with ASD, there are also alterations in the level of neurotransmitters produced in the basal ganglia. Paval's (2017) dopamine hypothesis of autism links social and communication deficits to lower levels of dopamine found in the mesocortical pathway of the basal ganglia, which connects the midbrain to the prefrontal cortex, while repetitive behaviours are explained by deficient levels of dopamine in the frontostriatal dopamine pathway, which connects the basal ganglia and frontal cortex. A post-mortem study reported increased levels of DRD2 expression (expression of receptors for dopamine in the indirect pathway) in autistic individuals compared to controls, suggesting there is an imbalance in indirect pathway functioning, which may partially explain motor issues and repetitive behaviours in ASD (Brandenburg et al., 2020). According to Figure 2, this imbalance in indirect pathway functioning might be expected to result in proactive control changes. Lower levels of serotonin binding were found extensively throughout the brain in individuals with ASD, particularly in the anterior and posterior cingulate cortices, which was correlated significantly with social deficits and repetitive behaviours (Nakamura et al., 2010). Dopamine binding was also found to be significantly increased in the OFC (Nakamura et al., 2010).

### 1.7 Functional Changes of Cognitive Control in ASD

The alterations in brain structure and neurotransmitters may be a component connecting the deficits in cognitive control demonstrated in autism research, and could support the executive function hypothesis of autism. Cognitive flexibility has been extensively found as a deficit that is specific to children with ASD (Ozonoff et al., 1994,

Ozonoff & Strayer, 1997, Ozonoff & Jensen, 1999, Lopez et al., 2005). Deficits in response inhibition have also been identified in children with ASD (Geurts et al., 2014), and have been correlated with repetitive behaviours symptom severity (South et al., 2007, Mosconi et al., 2009). Individuals with ASD have shown impairments in completing tasks that required maintenance of task-relevant information and simultaneous inhibition of a prepotent response tendency (Solomon et al., 2008). These findings indicate deficits in multiple areas of cognitive control, and are quite heterogenous across different levels of the autism spectrum. Solomon et al. (2014) proposed that children with ASD were more likely to utilise reactive inhibition strategies for longer during maturing years than typically developing children, and this was correlated with performance on tasks measuring reinforcement learning. This provides further support that proactive inhibition develops with maturity (Luna et al., 2007, Braver, 2012), and highlights that children with ASD show deficits in developing this form of proactive control.

## 1.8 Assessment of Response Inhibition and Reinforcement Learning in ASD

### *1.8.1 Stop Signal Task*

Previous research has suggested that children with ASD showed significant deficits in response inhibition compared to neurotypical children (Geurts et al., 2004, Bishop & Norbury, 2005, Christ et al., 2007, Lemon et al., 2011, Leno et al., 2018), with deficits in task performance correlating positively with autistic symptom severity. These studies measured response inhibition using various neuropsychological tasks that measure multiple facets of cognitive control simultaneously and do not differentiate between proactive and reactive control (e.g., the Stroop Task) which may have impacted potential findings in this area (Geurts et al., 2014). Studies utilising the SST, which can measure reactive inhibition (SSRT) more precisely, have extensively identified no significant differences in SSRT between

children with ASD and neurotypical children (Ozonoff & Strayer, 1997, Adams & Jarrold, 2012, Schmitt et al., 2018, Gooskens et al., 2019, Albajara Sáenz et al., 2020), suggesting there is no significant deficit in reactive inhibition specifically. In comparison to other developmental disorders, a meta-analysis found significant heterogeneity in SSRT scores in individuals with ASD, suggesting this deficit is not as large a component of ASD in comparison with individuals with ADHD, who tended to show homogenous deficits (Lipsyzc & Schachar, 2010). This heterogeneity may be a potential indicator as to why there are both significant and non-significant findings across research in the autism population, and highlight how this disorder is distinct from other disorders that present in childhood.

A recent study utilised the SST to measure both proactive and reactive inhibition and found that children with ASD showed no significant difference in SSRT compared to neurotypical children, while significant deficits in proactive inhibition were found (Schmitt et al., 2018). These deficits were also correlated with stronger repetitive behaviours (Schmitt et al., 2018). Similar findings have been found in a study using an ocular response inhibition task (Kelly et al., 2020). These findings indicate that inhibitory deficits may lie in the proactive inhibition pathway between the basal ganglia and cortex, rather than the reactive inhibition pathway. This study aims to build upon these findings by using the reinforcement learning task as an additional measure of proactive inhibition, to determine whether proactive inhibition deficits are consistent across different measures.

### *1.8.2. Reinforcement Learning Task*

Currently, no research has been conducted into how individuals with ASD react during trials of high and low conflict on the reinforcement learning task. This thesis therefore aims to address this gap in the literature by exploring the relationship between autistic trait level and proactive inhibition assessed via a comparison of reaction time during high and low

conflict trials. Findings in Parkinson's Disease patients by Frank et al. (2007) suggest that high and low conflict trials in the probabilistic selection task involve alterations in the level of STN functioning, and that patients responded more impulsively on high conflict win-win trials, when their STN activity was suppressed via deep brain stimulation. Similar findings have been reported in Parkinson's Disease patients with deep brain stimulation on other tasks measuring proactive and reactive inhibition, with more impulsive responding found on Go/No Go tasks and the SST (Ballanger et al., 2009, Benis et al., 2014). Both ASD and Parkinson's Disease have deficits in frontostriatal circuitry (Schmitt et al., 2018), and can both present with repetitive behavioural symptoms that include impulsivity and ritualistic actions (Hollander et al., 2009). These behaviours are evident in Parkinson's Disease patients who are taking dopaminergic medication or undergoing deep brain stimulation, which alters the functioning of frontostriatal circuitry. With these similarities in mind, there is potential for similar impulsive responding to occur in individuals with ASD during high conflict trials on the reinforcement learning task.

### 1.9 Significance

Results across these studies demonstrate that there are significant deficits in cognitive control, and this is related to the type of symptoms expressed and symptom severity in individuals with ASD. Difficulty inhibiting prepotent responses may be responsible for restrictive and repetitive behaviours (Lopez et al., 2005, Solomon et al., 2009, Agam et al., 2010, D'Cruz et al., 2013, Schmitt et al., 2018), and deficits in proactive inhibition may clinically manifest as an inability to use contextual cues to stop performing an action (e.g., talking about a topic of interest when it is no longer appropriate) (Mirabella, 2021). Alterations in frontostriatal circuitry may contribute to behavioural rigidity and motor symptoms (Brandenburg et al., 2020). Therefore, improving cognitive control could become

a potential target for intervention in the future (D'Cruz et al., 2013, Kelly et al., 2020). Furthermore, the ability to identify specific deficits in executive functions (e.g., inhibition and cognitive flexibility) that are indicative of ASD may help to distinguish ASD in young children, as opposed to other disorders of executive function such as ADHD (Ozonoff & Strayer, 1997).

### 1.10 Thesis Aims

The purpose of this study is therefore to explore the level of proactive inhibition deficits in individuals with differing levels of autistic traits. Reactive inhibition will also be tested in order to compare between the two types of cognitive control. As the phenomenon of proactive control has rarely been studied, two measures of proactive inhibition will be utilised to get a more stable estimate of potential deficits in individuals with differing levels of autistic traits, which we will measure in our sample of adults from the general population. Our first hypothesis is that there will be greater levels of deficits in post-error slowing in the Stop Signal Task in individuals with higher scores on the AQ-28. Higher scores on the AQ-28 are indicative of higher levels of self-reported autistic traits, so we expect to see greater levels of deficit in post-error slowing in the SST in individuals with higher levels of self-reported autistic traits. Our second hypothesis is that there will be greater levels of deficits in reaction time slowing during high conflict trials in the reinforcement learning task in individuals with higher AQ-28 scores. Thirdly, we hypothesise that there will be no significant difference in reaction inhibition capabilities in our sample, regardless of AQ-28 scores. To summarise, the first two hypotheses test whether autistic traits are associated with weaker proactive inhibition, whereas the third hypothesis tests whether reactive inhibition is unrelated to autistic traits. Furthermore, exploratory analyses will be conducted isolating the Social Skills and Routine subscales of the AQ-28 testing these hypotheses, in order to see if

there are significant findings relating to the specific ASD diagnostic criteria (deficits in social communication and presence of repetitive behaviours). We expect to find greater deficits in proactive inhibition in individuals with higher scores on the Routine subscale of the AQ-28, as higher scores are more representative of repetitive behaviours in ASD.

# Method

## 2.1 Participants

The experimental protocol was approved by the University of Adelaide Human Research Ethics Committee and was administered in compliance with the Declaration of Helsinki (2013). A total number of 152 adults ($N$ = 111 females, mean age = 45.05 years, range = 18-81 years) participated in this study. All participants were recruited for a larger study conducted by the University of Adelaide which researched cognition and healthy ageing across the lifespan. Participants of the larger study were recruited via Gumtree and Facebook advertisements posted online. Seven participants were excluded from the results of this study due to inconsistency in SST measurement: four participants were removed due to omissions in Go trials during the SST (ranging from 34-64 trials), affecting the critical SSD algorithm calculation, two participants were removed due to excessive slowing during Go trials which subsequently affected critical SSD calculation, and one participant was removed due to only having three successful inhibitions on Stop trials, so critical SSD could not be calculated. Two more participants were further not included in the analyses, as they did not provide results on the AQ-28.

## 2.2 Procedure

Participants were required to complete the AQ-28 online before commencing the in-person assessment tasks. The SST and reinforcement learning tasks were completed in the laboratory on an iPad Pro. Participants were given headphones to listen to pre-recorded instructions (refer to Appendix A and B) about how to complete the tasks, with a short visual animation played before task commencement. Participants were able to replay instructions before task commencement or ask an experimenter for clarification of the instructions.

## 2.3 Materials

Autistic trait level was measured using the Autism-Spectrum Quotient-28 (AQ-28) developed by Hoekstra et al., (2011), which is a shortened version of Baron-Cohen et al.'s (2001) Autism Spectrum Quotient Scale. The purpose of the AQ-28 is to measure quantitative autistic traits in the general population in a brief, self-administered fashion for laboratory research purposes. The measure asks questions regarding social (e.g., 'I enjoy meeting new people') and non-social (e.g., 'I prefer to do things the same way over and over again') aspects of behaviour and cognition. The AQ-28 is reliably correlated with Baron-Cohen et al.'s (2001) original scale ($r$ = .93-.95), and differs in that it has a two main factor structure, namely a social behaviour factor and a fascination for patterns and numbers factor. The social behaviour factor is broken down into four lower-level factors, including social skills, routine, attention switching and imagination.

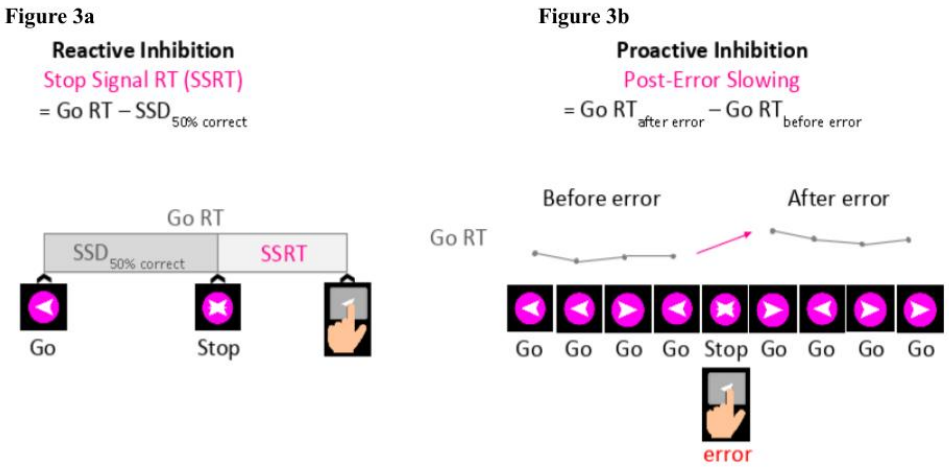
### *2.3.1 Stop Signal Task*

Proactive and reactive inhibition were measured using the Stop Signal Task (SST; Logan & Cowan, 1984) (see Table 1). Participants were presented with a blank screen with two arrow buttons pointing left and right, and instructed to press the arrow that corresponded to a Go stimulus that would appear between them as fast as they could, and suppress this action if the Stop signal appeared. In Go trials, the Go stimulus (depicted as a pink arrow) would appear after one second. Participants have one second to respond by pressing the button the Go stimulus was directing towards. If the participants made a response, the selected button would darken for 200ms, and Go reaction time would be measured. In Stop trials, the Go stimulus would be presented after one second, and after the SSD, a second arrow facing the opposite direction would be superimposed onto the Go stimulus. This superimposed arrow was the Stop signal. The SSD ranged between 50-500ms, and would be

updated before each Stop trial by the Bayesian algorithm developed by Livesey & Livesey (2016). The algorithm aims to identify which SSD leads to the correct withholding of a response on 50% of trials. This is referred to as the critical SSD, and is used to calculate the Stop Signal Reaction Time (SSRT). The SST consisted of 120 Go trials and 60 Stop trials. Half of the trials targeted the left arrow, and the other half targeted the right arrow, with arrow direction and Go/Stop trials randomised during the task. Reactive inhibition was measured by calculating the SSRT, which is the difference between the mean Go reaction time (Go RT) and the critical SSD (see Figure 3a). Proactive inhibition was measured by post-error slowing (see Figure 3b), and is calculated by subtracting the Go RT after an error from the Go RT before an error. Following Robertson et al., (1997), the Go RT before an error was calculated as the mean reaction time on Go trials within a four-trial window before each error, and the Go RT after an error was calculated as the mean reaction time on Go trials within a four-trial window after each error. Trials that fell both within a before-error window and an after-error window were omitted from calculations.

**Figure 3**

*Calculation of Reactive and Proactive Inhibition in the Stop Signal Task*



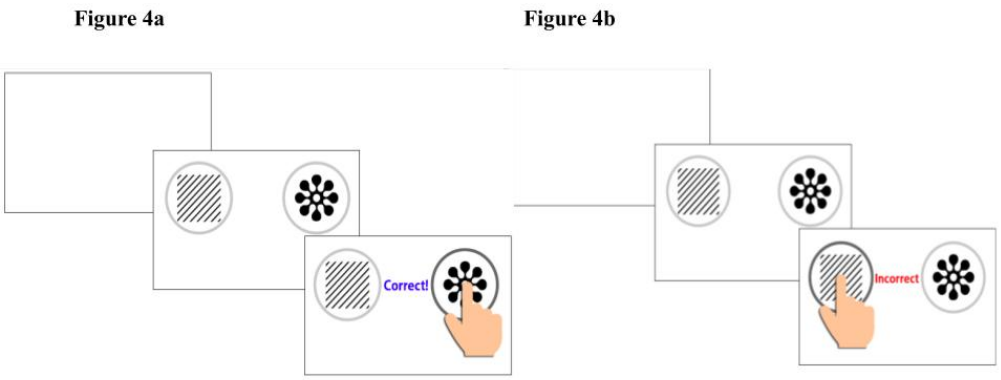
*Note:* Calculation of reactive and proactive inhibition during the Stop Signal Task. Figure 3a demonstrates calculation of the Stop Signal Reaction Time (SSRT), by subtracting the critical Stop Signal Delay (SSD) from the mean Go reaction time (Go RT). Figure 3b demonstrates the calculation of post-error slowing, by subtracting the Go RT after an error is made on a Stop trial (i.e., failing to inhibit the Go response) from the Go RT before an error is made.

### *2.3.2 Reinforcement Learning Task*

Proactive inhibition was also measured in the reinforcement learning task (see Table 1), which was modelled on Frank et al.'s (2004) probabilistic selection task. The task involved six sets of trials with different cue pictures. Each set had two phases, namely the training phase and the testing phase. The training phase involved two pairs of stimuli being presented together, with each stimulus pair having a different probability of being a correct choice. Within pair AB (where letters denote different visual stimuli), A was correct on 100% of trials, and B on 0% of trials, while within pair CD, C was correct on 75% of trials and D was correct on 25% of trials. The instructions before the task commenced (refer to Appendix A) informed participants that they would see many trials on which they would be asked to make a choice between two stimuli, and that after their selection they would receive feedback on their choice. Participants had four seconds to make a choice, otherwise 'No response detected.' would be displayed in red text. If the participant made a selection within the time frame, the stimuli selected was highlighted for 300ms before feedback was displayed for one second (see Figure 4). During the training phase, participants completed 16 trials in total, eight with each pair, and each cue was presented on the left and right side for half of the trials.

**Figure 4**

*Examples of Correct and Incorrect Trials in the Reinforcement Learning Task*




*Note:* The selection between two stimuli presented in the reinforcement learning task results in positive or negative feedback depending on the selected stimuli's reinforcement schedule. Figure 4a shows that positive feedback was displayed as 'Correct!' in blue text, while Figure 4b shows that negative feedback was displayed as 'Incorrect.' in red text.

During the test phase, participants were instructed to select the stimulus that 'feel most correct' based on what they learned in the training phase. Novel pairs of stimuli were presented, and participants would continue to make selections of the various stimuli. During the test phase, stimuli would be presented for four seconds, but after stimulus selection, no feedback was provided on their choice. In the testing phase, novel pairs of stimuli were presented (e.g., AC, AD, BC, BD) over 16 trials, with each pair being presented four times. All trials were randomised within each phase and each stimulus was presented on the left and right sides an equal number of times.

Reaction time was measured from the point of stimulus presentation to the selection of the stimulus by the participant. Test trials were categorised as high or low conflict trials depending on the difference in percentage of a stimulus being correct. Low conflict trials included AD and BC pairings (in which paired stimuli with discernibly different reinforcement probabilities), while high conflict trials consisted of AC and BD trials (in which paired stimuli had similar reinforcement probabilities). The procedure was then repeated another 5 times with different stimulus pictures, yielding a total of 48 low conflict test trials and 48 high conflict test trials that were averaged across the six sets. Proactive inhibition was calculated as the reaction time difference between high and low conflict test trials.

## Results

### 3.1 Descriptive Statistics

Table 2 shows the means, standard deviations and ranges for the individual differences on the AQ-28, including the overall score and separate subscales, and measures on the SST and reinforcement learning task used in the primary analyses. Calculations on the AQ-28 indicate that our sample was normally distributed and around the expected mean AQ-28 score range (though close to the higher boundary), with previous studies identifying average scores between 52-60 in multiple populations (Hoekstra et al., 2011).

**Table 2**

*Summary of Descriptive Statistics*

| Variable | Mean | Standard Deviation | Range |
|---|---|---|---|
| **AQ-28** | | | |
| AQ-28 Overall | 61.36 | 10.52 | 40 - 94 |
| AQ-28 Social Skills | 15.50 | 4.46 | 7 - 28 |
| AQ-28 Routine | 9.32 | 2.60 | 4 - 16 |
| AQ-28 Attention Switching | 9.21 | 2.34 | 4 - 16 |
| AQ-28 Imagination | 15.56 | 4.17 | 8 - 26 |
| AQ-28 Pattern Recognition | 11.76 | 3.37 | 5 - 20 |
| **Stop Signal Task** | | | |
| RT Before an Error (ms) | 575.45 | 92.57 | 374.80 - 784.30 |
| RT After an Error (ms) | 618.61 | 100.19 | 400.50 - 845.40 |

| | | | |
|---|---|---|---|
| Post-Error Slowing (ms) | 43.15 | 45.66 | -74.32 - 192.82 |
| Critical SSD (ms) | 357.73 | 127.42 | 75.06 - 549.999 |
| SSRT (ms) | 242.57 | 60.63 | 105.90 - 423.80 |
| **Reinforcement Learning Task** | | | |
| Low Conflict Trial RT (ms) | 1207.31 | 275.05 | 607.10 - 1958.60 |
| High Conflict Trial RT (ms) | 1376.37 | 301.48 | 557.60 - 2096.20 |
| RT Difference High vs Low Conflict (ms) | 169.06 | 165.28 | -218.58 - 623.21 |

*Notes:* Scores on the AQ-28 range from 28-112, with clinical cut-off for high-functioning autism scoring >70 (Hoekstra et al., 2011). The Social Skills subscale was comprised of 7 questions (4-28 total range), Routine and Attention Switching subscales were comprised of 4 questions (4-16 total range), the Imagination subscale was comprised of 8 questions (4-32 total range) and the Pattern Recognition subscale was comprised of 5 questions (4-20 total range). The SSD range was set between 5-550ms.

### 3.2 Post-Error Slowing

In order to determine whether proactive inhibition occurred in the study's population, a series of t-tests were conducted for the SST and reinforcement learning task and the study population. Post-error slowing was found to occur in the SST ($t(144) = 11.485$, $p = <0.001$, 95% CI = [35.93-50.87ms]). Reaction time slowing also occurred in high conflict trials

relative to low conflict trials in the reinforcement learning task ($t(144) = 12.317$, $p = <0.001$; 95% CI = [141.93-196.19ms]). These results indicate that proactive inhibition occurred in both tasks. Participants had greater reaction time slowing during the reinforcement learning task, and this may have occurred due to the task utilising top-down processing (comparing two stimuli with different reinforcement histories), compared to the relatively simple single motor response measured in the SST.

### 3.3 Proactive Inhibition Measures

The original intention when assessing proactive inhibition across the two measures was to create a single proactive inhibition factor to be used in the regression models, by conducting a principal component analysis with the post-error slowing scores on the SST and the reaction time difference between high and low conflict trials on the reinforcement learning task. Proactive inhibition measures on both tasks were weakly correlated ($r = -.04$), which did not justify conducting the principal component analysis. This correlation indicates that proactive inhibition is a multifaceted form of cognitive control and the two tasks were measuring proactive inhibition from different perspectives, namely as a top-down cognitive control process, and a single motor response process. For this reason, the two proactive inhibition measures from both tasks were kept separate in the regression analyses.

### 3.4 Regression Analyses

Regression models were used to assess the predictive relationship between AQ-28 scores and the measures of reactive and proactive inhibition in the SST and reinforcement learning task (see Table 3). The regression models adjusted for age and sex in order to examine whether our measures of proactive and reactive inhibition could predict AQ-28 scores above and beyond these potentially confounding variables. The Social Skills and

Routine subscales were also assessed separately (see Table 4 and 5) as exploratory analyses, due to the relationship between these subscales and the criteria for ASD diagnosis (deficits in social communication and presence of restrictive and repetitive behaviours). The Social Skills subscale is representative of the deficits in social communication, while the Routine subscale is representative of the repetitive behavioural component of ASD. Greater deficits were expected to be seen in the Routine subscale results compared to the Social Skills subscale, due to the hypothesised involvement of proactive inhibition in the presentation of repetitive behaviour.

**Table 3**

*Regression Model for AQ-28 Overall Scores*

| **Variables** | **B** | **Standard Error** | ***t*-value** | ***p*-value** |
|---|---|---|---|---|
| Intercept | 67.274 | 4.371 | 15.392 | <0.001*** |
| Age | -0.041 | 0.051 | -0.805 | 0.422 |
| Sex (Male) | 1.664 | 2.015 | 0.826 | 0.410 |
| SST – SSRT | -0.016 | 0.016 | -1.005 | 0.317 |
| SST – Post-Error Slowing | 0.004 | 0.019 | 0.238 | 0.812 |
| RL Task – High vs Low Conflict | -0.004 | 0.005 | -0.800 | 0.425 |

*Note*: SST = Stop Signal Task, RL Task = Reinforcement Learning Task, RT = reaction time.

The first regression model did not account for a significant proportion of variance, $R^2 = 0.031$, $F(5, 137) = 0.889$, $p = 0.490$, indicating that participant's overall scores on the AQ-28 was not predicted by deficits in either reactive or proactive inhibition, even when age and

sex were accounted for. These results do not support the first two hypotheses, according to which we expected lower proactive inhibition scores on both the SST and reinforcement learning task in participants with higher AQ-28 scores. The third hypothesis is supported, as SSRT did not predict the AQ-28 scores.

**Table 4**

*Regression Model for AQ-28 Social Skills Scores*

| **Variables** | **B** | **Standard Error** | ***t*-value** | ***p*-value** |
|---|---|---|---|---|
| Intercept | 15.483 | 1.839 | 8.418 | <0.001*** |
| Age | 0.017 | 0.021 | 0.786 | 0.433 |
| Sex (Male) | -1.378 | 0.848 | -1.625 | 0.106 |
| SST – SSRT | -0.001 | 0.007 | -0.187 | 0.852 |
| SST – Post-Error Slowing | 0.010 | 0.008 | 1.213 | 0.227 |
| RL Task – High vs Low Conflict | -0.003 | 0.002 | -1.287 | 0.200 |

*Notes*: SST = Stop Signal Task, RL Task = Reinforcement Learning Task, RT = reaction time.

The second regression model, which tested whether the Social Skills subscale of the AQ-28 could be predicted by deficits in reactive and proactive inhibition was also not significant, $R^2 = 0.044$, $F(5, 137) = 1.263$, $p = 0.284$. This model also does not support the first two hypotheses of this study with regard to deficits in proactive inhibition across the SST and reinforcement learning task, while showing support for the third hypothesis regarding no relationship between Social Skills scores and reactive inhibition.

**Table 5**

*Regression Model for AQ-28 Routine Scores*

| **Variables** | **B** | **Standard Error** | ***t*-value** | ***p*-value** |
|---|---|---|---|---|
| Intercept | 12.745 | 1.010 | 12.611 | <0.001*** |
| Age | -0.029 | 0.012 | -2.508 | 0.013* |
| Sex (Male) | 0.040 | 0.466 | 0.088 | 0.930 |
| SST – SSRT | -0.008 | 0.003 | -2.171 | 0.032* |
| SST – Post-Error Slowing | 0.004 | 0.004 | 1.043 | 0.299 |
| RL Task – High vs Low Conflict | -0.002 | 0.001 | -1.443 | 0.151 |

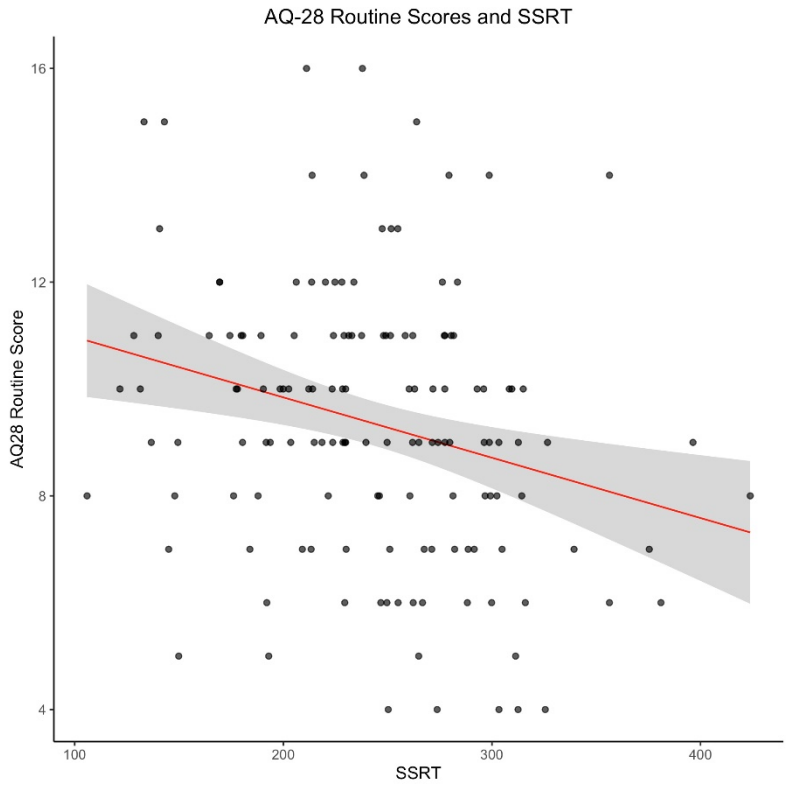
*Note*: SST = Stop Signal Task, RL Task = Reinforcement Learning Task, RT = reaction time.

The third regression model accounted for a significant proportion of variance in the Routine Subscale scores, $R^2 = 0.139$, $F(5, 137) = 4.443$, $p = 0.001$. The results from this model indicate that younger participants were more likely to score higher on the Routine subscale, and that higher Routine subscale scores were also significantly correlated with shorter SSRT duration, which indicates better reactive inhibition. Figure 5 shows this negative relationship between Routine subscale score and SSRT. This result does not support the third hypothesis, which stated that there would be no significant difference in SSRT duration, regardless of trait level. This indicates that lower scores on the Routine subscale were indicative of longer SSRT duration, meaning greater SSRT deficits were found in participants with lower Routine subscale scores. This result was surprising since previous studies that did report a relationship between ASD tendencies and proactive or reactive

inhibition always reported that these tendencies are associated with a deficit in response inhibition.

**Figure 5**

*AQ-28 Routine Scores and SSRT Duration*




*Notes:* The relationship between AQ-28 Routine subscale score and duration of SSRT. This figure illustrates the negative relationship between these two variables, where higher scores on the Routine subscale are correlated with shorter SSRT, indicative of better reactive inhibition.

A post-hoc analysis was conducted to explore this result further. The vast majority of research in ASD has been conducted in populations of children and adolescents, whereas we tested an adult population, including older individuals. It is possible that the relationship between inhibition and ASD tendencies changes with age, given the age-related effects on reactive inhibition, especially the decline typically seen in old age. To test for this possibility, we ran a post-hoc regression model that included the same predictors as the previous model,

but also tested the interaction between age and SSRT as a predictor of scores on the Routine subscale of the AQ-28 (see Table 6). The model accounted for a significant proportion of variance, $R^2 = 0.116$, $F(6, 136) = 4.274$, $p = 0.001$, with the interaction between age and SSRT duration predicting Routine subscale score approaching significance. In order to illustrate this finding, a median split was created to separate the participants into older and younger categories (above and below median age = 47). Figure 6 shows that the relationship between Routine subscale score and SSRT was not present in the younger cohort, but was present in the older cohort. This indicates that older individuals who have lower Routine subscale scores on the AQ-28 are more likely to have greater SSRT duration (i.e., worse reactive inhibition).

**Table 6**

*Post-Hoc Analysis: Interaction Between SSRT and Age Predicting AQ-28 Routine Scores*

| **Variables** | **B** | **Standard Error** | ***t*-value** | ***p*-value** |
|---|---|---|---|---|
| Intercept | 8.892 | 2.411 | 3.688 | <0.001*** |
| Age | 0.056 | 0.050 | 1.118 | 0.265 |
| Sex (Male) | 0.004 | 0.463 | 0.009 | 0.993 |
| SST – SSRT | 0.008 | 0.009 | 0.818 | 0.415 |
| SST – Post-Error Slowing | 0.005 | 0.004 | 1.065 | 0.289 |
| RL Task – High vs Low Conflict | -0.002 | 0.001 | -1.556 | 0.119 |
| Age*SST - SSRT | <0.001 | <0.001 | -1.757 | 0.081 . |

*Note*: SST = Stop Signal Task, RL Task = Reinforcement Learning Task, RT = reaction time.

**Figure 6**

*Comparison Between Age Groups on AQ-28 Routine Score and SSRT Length*


*Notes:* Age group comparison on AQ-28 Routine subscale score and duration of SSRT. While participants in the younger cohort did not show a relationship between SSRT length and Routine subscale score, participants in the older group who had lower Routine subscale scores tended to have longer SSRT duration. Note that age was analysed as a continuous variable and is plotted here as a binary variable for illustration purposes only.

# Discussion

## 4.1 Summary of Results

This study examined self-reported autistic trait level in members of the general population and measures of reactive and proactive inhibition, in order to explore the relationship between autistic trait level and deficits in cognitive control. The results did not find a significant relationship between autistic trait level measured on the AQ-28 and proactive inhibition performance on the SST or reinforcement learning task, which did not support the first two hypotheses of the study. The results indicated that proactive inhibition abilities remained intact in individuals with higher levels of autistic traits, which is inconsistent with several recent studies that utilised the SST (e.g., Schmitt et al., 2018, Schmitt et al., 2020, Kelly et al., 2020). These non-significant findings remained when conducting exploratory regression analyses using the Social Skills and Routine subscales, which were used as proxies for ASD symptomatology (deficits in social communication and the presence of restrictive and repetitive behaviours). Overall, these findings suggest that proactive inhibition pathways remain functional in individuals with sub-clinical higher levels of autistic traits, which subsequently means that there are no significant deficits in proactive control processes in the study's population, regardless of autistic trait level.

The results from this study also found no significant differences between autistic trait level and SSRT duration, showing support for the third hypothesis, which posited that reactive inhibition abilities were unrelated to autistic trait level. The non-significant results for the SSRT duration and overall AQ-28 scores are consistent with previous findings in SST studies conducted in populations of children with ASD (e.g., Ozonoff & Strayer, 1997, Adams & Jarrold, 2012, Schmitt et al., 2018, Gooskens et al., 2019m Albajara Sáenz et al., 2020). These findings show further support for there being no significant deficits in reactive

control processes in this population. To justify these non-significant findings, a post-hoc power analysis was conducted to ensure that this study had adequate power. The study's population as quite large, meaning that very small effect sizes should be able to be found. With a given power of .8, $a = .05$, five predictors and our sample size of 152, effect sizes as small as $f^2 = 0.08$ would have been able to detected.

After conducting exploratory analyses to account for the Social Skills and Routine subscales on the AQ-28, no significant relationship was found between SSRT and scores on the Social Skills subscale, providing further support for the third hypothesis. However, a significant relationship was found between SSRT and Routine subscale score, which did not support the third hypothesis. After conducting further analyses to understand the nature of this significant relationship, an interaction between age and Routine subscale score on the AQ-28 modestly predicted SSRT duration, with older participants with lower Routine subscale scores having longer SSRT duration, indicating greater deficits in reactive inhibition abilities. While age-related lengthening of SSRTs has been found previously (e.g., Smittenaar et al., 2015), the negative relationship between reported autistic trait level and SSRT duration has not been found previously in the literature, and is counter to previous results that found reactive inhibition deficits in children with ASD (e.g., Geurts et al., 2004, Bishop & Norbury, 2005, Christ et al., 2007, Lemon et al., 2011, Leno et al., 2018). Overall, this finding suggests that there are age-related changes in the reactive inhibition pathway, leading to lengthening of SSRT and greater deficits in reactive control processes, which could result in a more complex relationship with autistic traits.

The study's findings will be discussed further as to how they add to previous research on proactive control in ASD, in relation to the executive function hypothesis of ASD, and

age-related changes in reactive and proactive control. Strengths and limitations of the study will be explored, as well as future considerations for research in this area.

### 4.2 Cognitive Control in ASD

This study's results did not find any significant differences in both reactive and proactive control abilities in relation to overall AQ-28 scores. While non-significant differences in reactive inhibition responses have been found previously in ASD populations (e.g., Ozonoff & Strayer, 1997, Adams & Jarrold, 2012, Schmitt et al., 2018, Gooskens et al., 2019, Albajara Sáenz et al., 2020), this study conflicts with growing findings of proactive inhibition deficits in individuals with ASD (e.g., Schmitt et al., 2018). This study did not find significant differences in proactive inhibition abilities on multiple measures, indicating that proactive control abilities remained consistent across task performance in the study's population, and were not significantly influenced by autistic trait level. As exploration into proactive inhibition abilities in the ASD population is still a new area of investigation, these results may indicate that proactive inhibition deficits are not evident in the general population with regard to self-reported autistic traits, but may still potentially be evident in clinical populations. Much akin to the conflicting findings in reactive inhibition research, there may be more studies in the future that find no significant deficits in proactive inhibition, as ASD is a disorder that has significant heterogeneity in symptom presentation. Put differently, there may be individuals with ASD who do have significant deficits in proactive inhibition, but this may not be a significant finding across the entire ASD population (Geurts et al., 2014). The inconsistency of findings in response inhibition as a component of cognitive control may indicate that deficits in response inhibition alone are not enough to be used as an endophenotype for repetitive behaviours in ASD (Geurts et al., 2014). Furthermore, this study's population was comprised of adults, while previous studies measuring deficits in

proactive inhibition had samples of children and adolescents. As proactive inhibition abilities develop later than reactive inhibition abilities, typically from the age of 15 and remain at a constant level throughout adulthood (Smittenaar et al., 2015), we may have not seen differences in this population due to proactive control being fully developed already. Further research is needed in the adult ASD population to investigate whether deficits remain after proactive control abilities are fully developed.

### 4.3 Executive Function Hypothesis of ASD

The executive function hypothesis of ASD posits that deficits in cognitive control (e.g., response inhibition) are central to the presence and severity of autistic symptoms. The hypothesis gained attention as it attempted to provide an explanation for the presence of repetitive behaviours in ASD (Pennington & Ozonoff, 1996). This study based its proposed hypotheses on this approach and in relation to previous findings, aiming to investigate whether there are significant differences in proactive and reactive control in the study's population. These findings suggest that overall scores on the AQ-28 are not indicative of significant deficits in either proactive or reactive control abilities, and these results contradict with the overarching executive function hypothesis of ASD. Several weaknesses of the executive function hypothesis have been posited previously when considering previous research results finding no significant deficits in response inhibition (e.g., Lopez et al., 2005). The hypothesis is framed as a deficit model which means it would not have predicted that intact cognitive control processes (e.g., response inhibition) could be significantly related to the presence of repetitive behaviours (Lopez et al., 2005). It has also been proposed that no single cognitive control process (e.g., response inhibition) can account for the presence of restricted and repetitive behaviours in ASD entirely (Lopez et al., 2005). Different subsets of ASD may involve differing degrees of deficit, implying that one cognitive theory may not be

able to explain all symptoms, leading to large individual differences when measured on different tasks (Geurts et al., 2014). This study's findings build upon these criticisms, as the results did not find significant levels of deficit on SST and reinforcement learning task performance that were correlated with higher levels of self-reported autistic traits. While the executive function hypothesis of ASD is able to explain some cognitive differences in individuals with ASD compared to the general population, it may not be an appropriate model to explain varying levels of ASD-like traits found in the general population at subclinical levels.

### 4.4 Age Related Changes in Reactive Control

The results from this study found an age-related lengthening of SSRT duration, indicating deficits in reactive inhibition in older participants. This finding suggests that reactive control abilities do show decline throughout adulthood, and is consistent with findings in reactive inhibition research that has been conducted across the lifespan in the general population. Previous research exploring reactive inhibition abilities across the lifespan have found that reactive inhibition abilities increase during childhood as the brain develops, demonstrated through decreasing SSRT duration (Williams et al., 1999, Bedard et al., 2002, Tillman et al., 2008). Subsequently, reactive inhibition abilities then decrease with age, demonstrated by SSRT lengthening, especially during old age (Williams et al., 1999, Bedard et al., 2002, Bloemendaal et al., 2016). This may be potentially due to reduced frontal lobe integrity (Kramer et al., 2004). A study using diffusion weight imaging correlated lengthening of SSRT during SST performance in older age participants to structural decline of STN projections, particularly in connections between the pre-SMA and STN (Coxon et al., 2012), which are both components of the hyperdirect pathway (Aron, 2011, Jahanshahi et al., 2015). Together, these findings suggest that age-related decline in reactive inhibition abilities

may be caused by neurobiological changes in the brain that occur in old age, such as significant decreases in connectivity between the frontal lobe and basal ganglia.

Alterations in neurotransmitter levels may also be involved in the decline in reactive inhibition abilities in older adults. Post-mortem studies have found that dopamine neuron loss in the basal ganglia occurs at a rate of 5-10% per decade (Fearnley & Lees, 1991, Ma et al., 1999). To counter this loss in production, the brain attempts to boost dopamine synthesis in the remaining neurons, which has been correlated with non-optimal functioning in the basal ganglia and impacted performance on tasks involving the frontal lobe (Braskie et al., 2008, Klostermann et al., 2012). Research into dopamine's role in response inhibition and other cognitive control processes has proposed an inverted U-shaped function of performance, in which individuals with too little or too much dopamine production perform sub-optimally on cognitive control measures (Frank et al. 2007, Akbari Chermahini & Hommel, 2009, Colzato et al., 2009). Studies of both individuals with conditions that affect dopamine production such as Parkinson's Disease (van den Wildenberg et al., 2006) and healthy populations (Colzato et al., 2009) have found increased SSRT duration while completing the SST when dopamine levels are either too high or too low. While research into this inverted U-shape function of performance has not been explored in populations with ASD, there may be potential for similar results to be seen in this population due to dopamine signaling abnormalities in the mesocortical and frontostriatal pathways that contribute to ASD (Paval, 2017). This phenomenon may also provide a potential explanation as to why age-related differences in SSRT were predictive of scores on the Routine subscale of the AQ-28. Individual differences in performing Routine behaviours (e.g., preferring to perform activities in the same manner, presence of negative feelings if their daily routine in disturbed) may arise from increased levels of dopamine production when performing these behaviours (Paval, 2017). As this study found that older individuals with lower scores on the Routine subscale had longer

SSRT duration, this may indicate that individuals who have greater preference for routine living may be producing dopamine closer to optimal levels in the U-shaped performance function curve, while older individuals who have lower Routine scores may experience sub-optimal dopamine production, leading to longer SSRTs. To summarise, this study's findings add to the literature by showing support for age-related decline in reactive control processes, as demonstrated by lengthening SSRT during the SST, and this may have occurred through neurobiological changes in the connections between the frontal lobe and basal ganglia, or alterations in dopamine production that occur in old age. Scores on the Routine subscale of the AQ-28 may be related to individual differences in dopamine production, leading to changes in SSRT duration in older participants.

### 4.5 Age Related Changes in Proactive Control

This study found no significant age-related changes in proactive inhibition abilities. This finding supports previous results from Smittenaar et al.'s research (2015), which found that proactive inhibition abilities remained consistent throughout the lifespan after proactive control abilities were fully developed. The development of proactive inhibition capabilities occurs during adolescence (Luna et al. 2007, Vink et al., 2014), as functional connectivity increases between the frontal lobe and the basal ganglia (Vink et al., 2014). Unlike Smittenaar et al.'s study (2015), which found women having greater proactive inhibition abilities across all ages, no significant gender associations between age and proactive inhibition abilities were found in this study.

In older adults, other studies have found results that conflict Smittenaar et al.'s (2015) findings, as they found impaired proactive inhibition skills in older adults, but only in circumstances with high information load, leading to reduced preparation capacity (van der Laar et al., 2011, Bloemendaal et al., 2016). Proactive slowing in these situations has been

described as a cautious behaviour due to response patterns favouring accuracy over speed during SST trials (Starns & Ratcliff, 2010). Under high information load trials, older adults were not able to effectively utilise this strategy, leading to an inability to maintain these proactive slowing abilities (Bloemendaal et al., 2016). Interpreting these findings together, cognitive ageing and information load have inverse relationships on proactive control abilities. While proactive control tends to increase during older age to avoid engaging in reactive inhibition processes (van der Laar et al., 2011, Bloemendaal et al., 2016) (which tend to show age-related decline), high information loads tend to engage more reactive inhibition processes and resulting in decreased length of proactive inhibition (Bloemendaal et al., 2016). This study's SST did not have altered information loading conditions, so the task would be equivalent to the low information load condition in Bloemendaal et al.'s (2016) study. This study's findings are consistent with this research, as we no significant differences between younger and older participants on measures of proactive inhibition on the SST or reinforcement learning task were found, both of which had low information load. This may also be in part due to the instructions given to participants in multiple formats, including written, verbal and visual animations, reinforcing the need to respond as fast as they can in both tasks, as clear information about actions that may need to be stopped has been found to improve both speed and selectivity of inhibition (Smittenaar et al., 2013). Research into proactive inhibition is still needed, and so the findings from this study provide support for proactive control being an ability that does not significantly change over the lifespan. While there are conflicting results in this area, this may be due to the involvement of other variables, such as information loading.

### 4.6 Strengths

A strength of this study lies in the use of multiple measures of proactive inhibition. Earlier studies in this area have utilised the SST effectively to measure proactive inhibition (e.g., Schmitt et al., 2018), but have not included other measures to provide more stable estimates of proactive control from behaviour. This study is also the first to utilise high conflict trial reaction time as a measure of proactive inhibition in the context of ASD, as this measure has only been explored in the context of Parkinson's Disease so far (e.g., Frank et al., 2007). While no significant differences in proactive inhibition were found in the study's population, the results demonstrated that proactive inhibition occurred during the high conflict trials of the reinforcement learning task, indicating this is a suitable measurement technique in the context of autistic trait research. A second strength of this study is the age of the population. The vast majority of research into ASD and cognitive control deficits have been conducted in populations of young children and adolescents. While ASD is a lifelong condition, research into the effects of maturation and ageing in adulthood and cognitive control performance in the ASD population have not yet been explored thoroughly. The study's sample had a wide age range from early adulthood into old age, giving us the opportunity to assess age-related changes in proactive and reactive control abilities in relation to autistic trait levels in adulthood.

### 4.7 Limitations

This study also has several limitations. Firstly, autistic traits were measured, relying on subjective self-reporting, which assumes that participants are introspective enough to report accurately on their own behaviour. The measurement of autistic traits also provides information about how individuals behave in their daily lives in the context of ASD symptomatology, but the results on the measure do not explain why people behave in this

manner, or whether these behaviours are clinically relevant or akin to repetitive behaviours evident in individuals with ASD. Secondly, Hoekstra et al. (2011) suggests scores >70 are correlated with high-functioning autism categorisation for research purposes, but this scale is not used as a diagnostic measure, thus scores on the AQ-28 cannot be used as a diagnostic measure of ASD in this sample. This limits the ability to generalise the results to the ASD population. Thirdly, the use of the Routine subscale of the AQ-28 is limited in terms of its ability to accurately be representative of repetitive behaviours, due to the subscale's length. As the Routine subscale is only comprised of four questions, this subscale may not capture the scope of restrictive and repetitive behaviour practices in the study population. The limited number of questions may also not cover the scope of clinically relevant repetitive behaviours that are performed in individuals with ASD, which also limits generalisability of the results to individuals with ASD.

### 4.8 Future Directions

Future research in this field of exploring proactive control should consider utilising multiple measures of proactive inhibition in order to find more stable estimates of proactive control from behaviour. As the performance on high conflict trials in the reinforcement learning task was significantly able to measure proactive inhibition in this study's sample, and has been previously utilised in Parkinson's Disease patient populations effectively, this may be a potential measure that could be utilised in ASD studies with clinical populations. By using multiple measures of proactive inhibition, future research will be able to explore the complex relationship between performance on measurement tasks and basal ganglia pathway activation and how this relates to the presence and severity of restrictive and repetitive behaviour in ASD. Furthermore, additional research is still needed to clarify the complex

nature of basal ganglia pathway functioning and presence of symptomatology in ASD across the adult lifespan.

Further cross-sectional studies are also needed to determine the complex relationship between age and development of reactive and proactive inhibition abilities in ASD populations, especially between late childhood and adolescence into early adulthood, as well as older age, where there are significant changes in reactive and proactive control abilities. By identifying significant developmental changes, and timings of these changes in abilities, there may be future potential to create and implement therapeutic interventions in order to manage more severe symptoms in individuals with ASD.

### 4.9 Conclusion

The current study explored the functioning of different types of cognitive control in individuals with differing levels of self-reported autistic traits. Based on the executive function hypothesis of ASD, which posits that deficits in cognitive control are central to the presence of autistic symptomatology, this study aimed to explore different levels of potential deficits in both proactive and reactive control using measures of response inhibition and reinforcement learning. This study's findings suggest that autistic trait level in subclinical participants was not predictive of deficits in either reactive or proactive control and also suggest an interaction with age, which should be investigated further, as the relationship between cognitive control and autistic traits may change with age. These results are inconsistent with the previous findings that suggest proactive control deficits are evident in individuals with ASD, as this study utilised multiple measures of proactive inhibition in order to ascertain a more stable estimate of the behaviour occurring, but found no significant deficits in individuals with higher levels of autistic traits on either task. These results also add to the current debate in the literature that has found conflicting findings regarding reactive

inhibition abilities in individuals with ASD, by showing no significant deficits in reactive inhibition abilities in individuals with higher overall AQ-28 scores. This study also adds to the limited research conducted in adult populations in the context of ASD. In conclusion, further research into cognitive control and ASD should explore development of proactive and reactive control through the use of multiple measures and throughout the lifespan in order to understand the complex relationship between cognitive control processes and presence of restrictive and repetitive behaviours.

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## Appendix A

## Reinforcement Learning Task Instructions Script

Initial task instructions (with an animation):

“In this task, you’ll be presented with different pairs of pictures. For every pair you’re presented with, you’ll need to tap one of the two pictures, like this. Once you do, you’ll find out whether your response was correct or incorrect. This feedback will help you make the right choices more often. You’ll only have 4 seconds to make a response, so don’t waste too much time making a decision.

Remember, your task is to discover which pictures are more likely to be correct, and to maximise how many correct choices you make. Tap the ‘Replay’ button to watch these instructions again, or tap the ‘Start’ button to begin.”

Instructions before each test phase:

"It’s time to test what you’ve learnt! During this set of trials you will NOT receive feedback (’Correct!’ or ’Incorrect’) to your responses. If you see new combinations of pictures, please choose the picture that ’feels’ more correct based on what you have learnt so far. If you’re not sure which one to pick, just go with your gut instinct. Please remember to continue responding even though you will no longer receive feedback. Tap the ‘Start’ button to begin."

Instructions before each new set:

"In the next phase of this task, you will be presented with entirely new pairs of pictures. On every trial you will have to choose one of the pictures by tapping it. Like before, you will be informed whether your response was correct or incorrect. Your task is to discover

which pictures are more likely to be correct and to maximise how many correct choices you make. Tap the ‘Start’ button to begin."

## Appendix B

## Stop Signal Instructions Script

"In this task you will see two buttons. Press the left arrow button if an arrow pointing left appears… or the right arrow button if an arrow pointing right appears. You should try to respond as quickly as you can, so keep your hands near the buttons. However, try your best not to respond when you see two overlapping arrows. Stopping a response can be difficult, so try not to get too frustrated if you sometimes can't do it. Tap the 'Replay' button to watch these instructions again, or tap the 'Start' button to begin."

# Appendix C

# Ethics Approval

RESEARCH SERVICES
OFFICE OF RESEARCH ETHICS, COMPLIANCE AND INTEGRITY
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Our reference 33465

07 February 2020

Dr Irina Baetu
Psychology

Dear Dr Baetu

**ETHICS APPROVAL No:** H-2020-017
**PROJECT TITLE:** Cognitive function across the lifespan

The ethics application for the above project has been reviewed by the Human Research Ethics Committee and is deemed to meet the requirements of the *National Statement on Ethical Conduct in Human Research 2007 (Updated 2018)*.

You are authorised to commence your research on: 07/02/2020
The ethics expiry date for this project is: 28/02/2023

**NAMED INVESTIGATORS:**

| | |
|---|---|
| Chief Investigator: | Dr Irina Baetu |
| Student - Postgraduate Doctorate by Research (PhD): | Miss Brittany Dorothy Amelia Child |
| Student - Postgraduate Doctorate by Research (PhD): | Ms Lauren Mary Heidenreich |
| Student - Postgraduate Doctorate by Research (PhD): | Mr Salvatore Simone Russo |
| Student - Postgraduate Doctorate by Research (PhD): | Mr Nathan Daniel Beu |
| Associate Investigator: | Associate Professor Lyndsey Collins-Praino |
| Associate Investigator: | Professor Nicholas Burns |
| Associate Investigator: | Dr Sarah Cohen-Woods |
| Associate Investigator: | Dr Ahmed Moustafa |

**CONDITIONS OF APPROVAL:** Thank you for addressing the feedback raised. The application submit on the 7th of February 2020 is approved.

Ethics approval is granted for three years and is subject to satisfactory annual reporting. The form titled

Annual Report on Project Status is to be used when reporting annual progress and project completion and can be downloaded at http://www.adelaide.edu.au/research-services/oreci/human/reporting/. Prior to expiry, ethics approval may be extended for a further period.

Participants in the study are to be given a copy of the information sheet and the signed consent form to retain. It is also a condition of approval that you immediately report anything which might warrant review of ethical approval including:

- serious or unexpected adverse effects on participants, previously unforeseen events which
- might affect continued ethical acceptability of the project, proposed changes to the protocol
- or project investigators; and the project is discontinued before the expected date of
- completion.

Yours sincerely,

Professor Paul Delfabbro
Convenor

The University of Adelaide